Number of children and later-life mortality among Finns born in 1938–50

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Abstract

We investigated whether the number of children was associated with later-life mortality among Finns born in 1938–50, and whether observed living conditions in childhood and adulthood, chronic conditions, fertility timing and unobserved characteristics common to siblings explained the association. We used a longitudinal 1950 census sample to estimate mortality at ages 50–72. All-cause mortality relative to those with two children is highest among childless men and women, and elevated among those with one child, independently of the observed confounders. Fixed-effect models, which control for unobserved characteristics shared by siblings, clearly support these findings among men. Cardiovascular mortality is higher among men with no, one, or at least four children than among those with two children. Living conditions in adulthood contribute to the association between the number of children and mortality to a greater extent than childhood background, and chronic conditions contribute to the excess mortality of the childless.

Running title: Number of children and mortality among Finns

Introduction

Despite a long tradition of research on the association between childbearing and post-reproductive mortality, empirical evidence is still inconclusive (Hurt et al. 2006; Grundy 2009). A few studies show a U-shaped or reverse J-shaped relationship between parity and mortality, indicating an increase in mortality among childless women and those with numerous children, whereas others report ambiguous results (Grundy and Tomassini 2005; Hurt et al. 2006; Henretta 2007; Grundy 2009; Grundy and Kravdal 2010; Tamakoshi et al. 2011; Barclay and Kolk 2015). Most previous studies on contemporary populations concern women, and evidence for men is scarce (Friedlander 1996; Hypponen et al. 2005; Grundy and Kravdal 2008; Jaffe et al. 2009; Hank 2010; Tamakoshi et al. 2011). Only two previous studies analyse cause-specific male mortality by the individual number of children (Grundy and Kravdal 2010; Barclay and Kolk 2015). As far as we are aware, no studies examine whether the association between the number of children and mortality in both men and women could be attributed to childhood living conditions and severe chronic conditions, although it is suggested that they affect both fertility and mortality (Patja et al. 2000; Hayward and Gorman 2004; Goodman and Koupil 2009; Rijken and Liefbroer 2009; Bundy et al. 2011; Crump et al. 2013; Elo et al. 2014; Nisen et al. 2014b). The few study designs that allow controlling for individuals' medical history are based on surveys enrolling relatively healthy subjects (Tamakoshi et al. 2011), and those with severe chronic conditions are excluded.

The first aim of our study was to examine the association between the number of children and all-cause and cause-specific mortality at ages 50–72, and to assess the extent to which observed living conditions in childhood, severe chronic conditions (e.g., intellectual disabilities, schizophrenia, and early disability retirement), living conditions in early adulthood, and the timing of the first child explain the association. We used a longitudinal sample from the 1950 census, linked to hospital discharge records and death records to analyse mortality among Finnish men and women born in 1938-50. The second aim was to assess whether unobserved social and genetic

confounders common to siblings explain the association with all-cause mortality. These fixed-effect models have previously been used in comparisons of Swedish siblings, and the findings suggest that parents with only one child have higher all-cause mortality than those with two children, regardless of various socio-economic confounders and gender (Torssander 2013). The present study investigates men and women separately, and incorporates the childless and the medical histories into the analyses.

Literature review

Mechanisms and selection

Having children is suggested to affect post-reproductive mortality in several ways (Kirkwood and Rose 1991; Doblhammer 2000; Grundy and Tomassini 2005; Hurt et al. 2006; Grundy and Kravdal 2010). The famous disposable soma theory refers to a trade-off between childbearing and female survival in the long term. According to the theory, less effort can be invested in the maintenance and repair of somatic tissues in the body because of the competing demands of reproduction with aging and decreased survival as a result (Kirkwood 1977; Kirkwood and Rose 1991). Evidence from studies on humans in contemporary societies does not clearly support the theory, however (Doblhammer 2000; Hurt et al. 2006; Grundy 2009). It is suggested that humans live in complex social and cultural settings that may largely offset any evolutionary trade-off between reproduction and survival (Doblhammer 2000). Moreover, women in contemporary populations may be less depleted after repeated pregnancies, because they are better nourished than women in historical populations (Hurt et al. 2006).

The most well-established evidence contradicting the disposable soma theory relates to certain hormone-related cancers in women, in that increasing parity appears to protect against breast cancer and cancers of the ovaries and uterus (Negri et al. 1988; Kvale et al. 1994; Clavel-

Chapelon and E3N-EPIC Group 2002; Collaborative Group on Hormonal Factors in Breast Cancer 2002; Britt et al. 2007; Grundy and Kravdal 2010).

Having children is suggested to have social consequences for the parents in addition to the biological consequences (Hurt et al. 2006). Mothers and fathers may enjoy social and financial support provided by their children, and may receive later-life care (Grundy and Tomassini 2005; Hurt et al. 2006; Grundy and Kravdal 2010). Child rearing may also provide an impetus to avoid health-damaging behaviour, and the health effects may become apparent later in life (Grundy and Kravdal 2008; Grundy and Kravdal 2010). The benefits of having several children may nevertheless be offset by the emotional and financial strains of child rearing. As several authors suggest, it would be useful to further compare the association between parenthood and later-life survival (or health) among women and men to determine whether social or pregnancy-related biological mechanisms predominate (Dekker and Schouten 1993; Kravdal 1995; Hurt et al. 2006; Jaffe et al. 2009; Grundy and Kravdal 2010).

The identification of social and biological mechanisms from observational data nevertheless remains challenging in that omitted factors may affect both childbearing and mortality. It is widely acknowledged that the association between the number of children and mortality among both men and women may be spurious in the sense that both are determined by the same characteristics, such as childhood background, health, marriage and socio-economic status. Hurt et al. (2006), for example, argue that the failure to adjust for socio-economic status could result in the overestimation of a negative association between high parity and survival, as poorer women tend to have more children and lower survival rates. In addition, failing to control for health status could result in the overestimation of a negative effect of childlessness in that severe health problems may decrease the likelihood of marriage or directly impair fertility. Although it is often argued that the health of a woman is more important in determining reproduction, it is possible that the role of

health selection is equally important among contemporary men and women, especially the selection influences of psychiatric disorders.

Many of the previous studies suffer from a lack of adequate control variables, especially those based on historical data (for a review see Hurt et al. 2006). Nevertheless, according to Doblhammer and Oeppen's (2003) historical study of the British peerage, formally taking health selection into account results in a trade-off between reproduction and later-life survival among women but not men.

Contradictory evidence among contemporary populations

It is suggested in studies conducted in contemporary modern societies that all-cause mortality among women with no children or only one child tends to be higher than among women with two children, independently of age, marital status and various demographic and socioeconomic confounders (Hurt et al. 2006; Grundy and Kravdal 2010). A few studies from England and Wales, Austria, Israel and Japan indicate that the mortality of mothers of at least four or five children is higher than that of mothers of two children (Doblhammer 2000; Manor et al. 2000; Grundy and Tomassini 2005; Tamakoshi et al. 2011). These results are consistent with the findings from new Swedish research indicating higher mortality among high-parity women born in 1915-1960 than among women with two or three children, regardless of the year of birth and various socio-economic confounders (Barclay and Kolk 2015).

On the other hand, recent studies from Norway show no elevated mortality among mothers of at least four or five children compared with women with two children. It is suggested that the health benefits of having many children may outweigh the costs in a family-friendly Nordic environment (Grundy and Kravdal 2008; Grundy and Kravdal 2010). Similarly, another Nordic study from Finland suggests that the mortality of women with five to nine children is somewhat lower as compared to the national average (Hinkula et al. 2006). The latter study does not examine

the risk of death by the individual number of births, however, and given the choice of an all-women reference group, the lower mortality of multiparous women may indirectly reflect the mortality disadvantage of those with no or only one child rather than the advantage of multiparity per se.

Despite the rapidly expanding interest in the association between the number of children and male mortality, empirical evidence from contemporary populations is still rarely available (Friedlander 1996; Hypponen et al. 2005; Grundy and Kravdal 2010; Hank 2010; Tamakoshi et al. 2011; Barclay and Kolk 2015). A study on a British cohort born in 1958 reports lower mortality among men with biological children than among all men when age, period and area of residence are controlled for (Hypponen et al. 2005). It is also reported in a Norwegian study that men with no children have the highest mortality, followed by men with only one child, regardless of age, year, education, marital status and regional characteristics. Men with two or three children have the lowest mortality (Grundy and Kravdal 2010). Similarly, studies on Swedish men report the lowest mortality among those with two or three children, net of various socio-economic confounders (Torssander 2013; Barclay and Kolk 2015). The Swedish studies did not control for marital status or the timing of the first child, however, which may have biased the results. A Japanese study restricted to healthy subjects, in turn, reports higher mortality among men with at least five, four, only one or no children than among those with two children, independently of age, marital status, education, employment status, region of residence, self-reported health history and various indicators of health behaviour (Tamakoshi et al. 2011). Other studies on contemporary male populations report no clear relationship between the number of children and mortality (Friedlander 1996; Hank 2010), and some are restricted to married men (Grundy and Tomassini 2006; Jaffe et al. 2009).

A few studies indicate that high numbers of offspring could be associated with the risk of cardiovascular diseases not only in women (Ness et al. 1993; Kvale et al. 1994), but also among men (Dekker and Schouten 1993; Grundy and Kravdal 2010). The association and its biological and

social mechanisms are still under debate (Dekker and Schouten 1993; Ness et al. 1995; Lawlor et al. 2003; Koski-Rahikkala et al. 2006; Hardy et al. 2007). Dekker and Schouten (1993) report in their study of Dutch civil servants and their spouses higher mortality attributable to coronary heart disease among married women with four or more children than among those with no children. Given the similar, although statistically non-significant, association among men, the authors suggest that both social and emotional factors and lifestyle, in addition to biological pregnancy-related factors, could contribute substantially to the association between high parity and cardiovascular diseases.

In the Norwegian context, Grundy and Kravdal (2010) report higher levels of mortality attributable to cardiovascular causes among men with no or only one child, and somewhat higher levels among those with at least four children, than among men with two children. Controlling for various demographic and socio-economic characteristics and the timing of the first child nevertheless rendered the small excess observed among fathers with many children non-significant. According to a recent Swedish study, men with numerous biological children are more prone to cardiovascular mortality than men with two children, independently of socio-economic confounders (Barclay and Kolk 2015). However, the latter study did not control for the early timing of the first child despite its documented association with both the number of children and male cardiovascular mortality (Grundy and Kravdal 2010).

Data and methods

Data sample

The present study was based on a 10-per-cent household sample (n=411,629) drawn from the 1950 Finnish census, with a detailed mortality follow-up from 1988 to 2010. We restricted the study sample to persons who were alive at the age of 50 and born in 1938-50 (n=87,573). The 1950 sample provided information on childhood and was linked to quinquennial censuses from 1970 to

1980 yielding information on early adulthood. The sample was further linked to hospital discharge records from 1969 to 2012 that provided information on chronic medical conditions and the dates of hospital admission. We also included household-based information on sibling relationships among the study subjects in 1950.

Statistics Finland carried out the data linkage using personal identification codes, which were introduced in the late 1960s. These codes are obtainable for approximately 94 per cent of men and 92 per cent of women born in 1938–50 and are included in the 1950 census sample (Statistics Finland 1997). Personal identification codes were missing for those who were lost to follow-up by the late 1960s – mainly due to death or emigration to Sweden. The use of the anonymised data sample was approved by Statistics Finland, the National Institute for Health and Welfare and the Social Insurance Institution (permissions TK-53-789-10, THL/1273/6.02.00/2013, KELA/40/522/2009).

Numbers of children

The analyses in this study cover men and women born in 1938–50, the aim being to construct almost complete records of the number of children ever born. The birth of a child has been registered in the files of a parent since the late 1960s using personal identification codes. A child born earlier is linked to a parent if the two lived together in the late 1960s. The fertility of women as observed in our data corresponds well with the levels reported earlier in Finland among corresponding female cohorts (Andersson et al. 2009). Appendix 1 gives the cohort-specific fertility rates of men and women in the 1950 census sample. Male fertility is only slightly lower than that of women, indicating that any bias attributable to unknown fatherhood is likely to be small. However, children who died before personal identification codes were introduced cannot be identified in the data. Adopted children are excluded.

Cause of death

The cause-of-death data is based on a harmonised international classification of diseases and related health problems (harmonised ICD-9, ICD-10) provided by Statistics Finland. The causes of death are further grouped into broader categories, including cardiovascular diseases, alcohol-related causes, accidents and violence, lung cancer, breast cancer, and other cancers. Cause-specific mortality is examined mainly in order to provide indirect evidence concerning lifestyle factors that may link the number of children and mortality.

Control variables

The year of birth

The year of birth was used as a control variable in all models, given that younger cohorts are known to have higher levels of childlessness and higher life expectancy (Andersson et al. 2009; Myrskylä 2010).

Childhood living conditions

Information on childhood living conditions originated from the 1950 census, at the time of which our study persons were 0–12 years old. We constructed childhood variables that measured the type of family, number of siblings, the occupation of the family head, the highest educational level of a parent, home ownership, housing conditions, and the region of residence at birth.

The type of family was categorised as follows: both parents, only a mother, only a father, and no parents. It was possible to identify the number of siblings within the household among those who lived with at least one parent. The few not living with either parent who were known to live in a household headed by a sibling or a step-sibling in 1950 were categorised as having one sibling. It was not possible to identify siblings who had already moved away from the family home or who were born after 1950. The occupation of the family head was categorised as

independent farmer, other self-employed or employer, professional or manager, and manual worker. If there was no information on the occupation of the family head we used information on the occupation of the head of the household. Parental education is that of the parent with the highest level of education. If neither parent was present, the lowest educational level was assumed. We also used home ownership, facilities such as central heating, piped water and sewerage, and the numbers of people per room to characterise housing conditions in childhood.

The region of residence at birth was categorised retrospectively according to the modern nomenclature of territorial units for statistics (NUTS level-3), with the exception of the Åland Islands, which were combined with Finland proper. The historical Finnish regions that were transferred to the Soviet Union after World War II were combined with other birth regions abroad. Table 1 shows the distribution of all childhood characteristics by gender and the number of children, excluding the region of residence at birth.

Chronic conditions

This study used three dichotomous indicators of chronic conditions, including schizophrenia, intellectual disability and early disability retirement. Individuals were categorised as having intellectual disabilities or schizophrenia if at least one hospital admission on these grounds was recorded between 1969 (at ages 18-30) and the day before reaching the age of 50. We used the principal diagnosis and three secondary diagnoses, which were based on the eighth (1969-1986), ninth (1987-1995) and tenth (1996-2012) revisions of the international classification of diseases, Finnish version [schizophrenia: ICD-8: 295, ICD-9: 295, ICD-10: F20; intellectual disability previously termed mental retardation: ICD-8: 310-315, ICD-9: 317-319, ICD-10: F70-F79] (Lääkintöhallitus 1969; Lääkintöhallitus 1986; STAKES 1999).

Intellectual disability and schizophrenia were used as control variables given that they are known to affect both fertility and mortality (Patja et al. 2000; Bundy et al. 2011; Crump et al.

2013), and their use in the analyses thus allows better control for health selection. Furthermore, the diagnostic criteria were relatively similar over time and across cohorts, and their prevalence among childless men and women was high enough to be shown (Table 2). However, prevalence among all parity groups is not shown on account of the small numbers and the privacy policy of Statistics Finland and the National Institute for Health and Welfare.

Substantial limitations in intellectual functioning and adaptive skills, most of which manifest immediately after birth or before adulthood, characterise intellectual disability (Das 2000; Skotko et al. 2009). Schizophrenia is a serious mental disorder that often involves illogical thoughts, atypical behaviour, and delusions or hallucinations. The onset of schizophrenic symptoms typically occurs in early adulthood between the ages of 18 and 30 (VandenBos 2000). Our hospital data do not permit identification of the onset of schizophrenic symptoms, but do indicate that 60 per cent of those diagnosed with schizophrenia in the two youngest cohorts (with the most complete medical history) had been admitted to hospital for mental health problems by the age of 28.

Schizophrenia is a complex mental disorder that results from the action of both genetic and environmental factors, many of which remain unknown (Murray et al. 2003). Studies have linked schizophrenia to prenatal and perinatal risk factors (Cannon et al. 2003), as well as to migration and urban residence (Boydell and Murray 2003). Although other social and psychological aetiological factors may also be important in developing schizophrenia (Boydell and Murray 2003), no prior evidence suggests that childlessness or childbearing is a risk factor for schizophrenia. Psychoses other than schizophrenia, such as mood disorders with psychotic features, were excluded in our study.

The individuals under study were categorised as having a disability pension if they were recorded by Statistics Finland as having retired or having been institutionalised by the age of 30-34 in 1970, 1975 or 1980, depending on the cohort. The reasons for retirement or institutionalisation are unavailable. However, official statistics show that the main reason for

retirement at the age of 30 in 1970, 1975 and 1980 was i) schizophrenia (icd-8: 295), followed by ii) moderate, severe and profound mental retardation (icd-8: 312-314), and iii) mild mental retardation (icd-8: 311). Approximately 60 per cent of all 30-year-olds on a disability pension were diagnosed with one of these conditions (derived from unpublished official statistics provided by the National Insurance Institution). The measure of premature retirement serves as a proxy for a medical condition assessed by medical doctors as chronic at an early stage of adult life.

Living conditions in early adulthood

We used six variables to characterise living conditions in early adulthood, including marital status, the region of residence and four indicators of socio-economic status. These characteristics were measured at the age of 30-34. Marital status was categorised as married, never married, divorced or separated, and widowed or unknown. It was controlled for because being non-married is associated with low parity in our study cohort, and marriage is known to be associated with lower mortality (Martikainen et al. 2005). We controlled for the region of residence because of its links with both fertility and mortality (Koskinen and Martelin 1997; Kulu et al. 2007). It was categorised according to the nomenclature of territorial units for statistics (NUTS level-3) with the exception of the Åland Islands, which were combined with Finland proper.

We controlled for four indicators of socio-economic status, including educational level, annual income, home ownership and the level of equipment in a dwelling, so as to reduce selection bias. There is evidence of an association between socio-economic status and both fertility and mortality (Elo and Preston 1996; Vikat 2004; Kravdal and Rindfuss 2008; Laaksonen et al. 2008; Tarkiainen et al. 2012; Nisen et al. 2014a). We used the following educational categories: basic or less, secondary and tertiary. Annual income was adjusted for changes in consumer prices, and categorised in euros for the year 2012. If no information was available (1.8 per cent of the sample), income was categorised according to the lowest level. In terms of equipment, dwellings

were categorised as well equipped, poorly equipped and unknown. A dwelling is considered well equipped if it has piped water, sewage connections, hot water, a flush toilet, washing facilities, and central or electric heating. Table 2 shows the distribution of all adulthood characteristics, excluding region of residence, by gender and the number of children.

The timing of the first child

The sample included month-level information on the time of birth of all the individuals under study and their children. These dates were set on the 15th day of the corresponding month and year to calculate the timing of the first child in completed years of the parent, grouped as follows: under 20 years, 20-24 years, 25-29 years, 30-34 years and 35 years and over. It was used as a control variable for the parents, as early-timed parenthood has been shown to increase post-reproductive mortality among both men and women (Grundy and Kravdal 2010).

Statistical methods

We used Cox proportional hazard models with age as the underlying time to estimate the risk of death. The follow-up started at the age of 50 (in 1988-2000 depending on the cohort) and ended on December 31, 2010, when those who were alive were aged between 60 and 72. The age of 50 was chosen as the starting point, the number of children presumably being complete by that age. In the cause-specific models, deaths due to causes other than that of interest were censored at the time of death.

The first aim of the study was to find out whether the association between the number of children and all-cause and cause-specific mortality persisted following controls for the confounding influences of observed characteristics (conventional Cox models). We first assessed the association controlling i) for the year of birth, and then sequentially for (ii) living conditions in childhood, (iii) chronic conditions, (iv) living conditions in early adulthood and (v) for the parents

the timing of the first child. All the control variables were added to the models as categorical, time-invariant variables. Parents with two children were chosen as the reference group. The results are reported as hazard ratios with 95-per-cent confidence intervals. The clustering in the data by household is taken into account in the estimation of standard errors.

We tested the assumption of proportional hazards by numbers of children using the Schoenfeld residuals obtained from the conventional Cox models (Tables 3 and 4, Models 4). The assumption held for most causes of death and parity groups. However, the evidence suggests that men with only one child have proportionally higher mortality attributable to lung cancer (p-value for proportional hazards < 0.001) and cardiovascular disease (p-value: 0.031) in midlife than at older ages, compared to those with two children. In contrast, an increase in alcohol-related mortality among fathers of one child and a decrease in cancer mortality among mothers of three children become apparent with advanced age (p-values: 0.045, 0.045). Appendix 4 shows the observed mortality rates by age group.

The second aim was to assess whether Cox models with sibling fixed effects would confirm the association between the number of children and all-cause mortality observed in conventional Cox models. Fixed-effect models allow controlling for unobserved characteristics shared by siblings. This is implemented in Stata software via a stratification option that allows different families to have different baseline hazard functions while constraining the hazard ratios to be the same across families. Hazard ratios cannot be estimated for variables that do not vary within a family, and therefore observed childhood characteristics were removed from these analyses. Each person is compared to his or her same-sex sibling, and only families in which deaths occur are included. On the basis of these 7,032 brothers and 3,283 sisters we estimated fixed-effects models. Fixed-effect models reduce the selection bias related to social and genetic characteristics common to siblings without the need to measure them directly. Despite the attractions of the fixed-effect Cox model, it also has disadvantages (Allison 2009). One of them is a substantial loss of statistical

power compared with conventional models. We therefore estimated fixed-effect sibling models only for all-cause mortality. In addition, unobserved characteristics, which siblings do not share may still induce bias in the estimates (Holmlund 2005; Lahey and D'Onofrio 2010).

Results

All-cause mortality among men

Among the men, the hazards of death relative to those with two children turns out to be highest for the childless (hazard ratio (HR) = 2.00, 95% confidence interval (CI): 1.88, 2.13), and the next highest among those with only one child and those with at least four children, regardless of the year of birth (1 child: HR = 1.28, 95% confidence interval (CI): 1.19, 1.37; 4+ children: HR = 1.17, 95% CI 1.07, 1.29). No elevated mortality is apparent among those with three children (Table 3).

Controlling for observed living conditions in childhood has a modest impact on the mortality estimates. Approximately three per cent of the excess mortality among childless men and eleven per cent among men with only one child relates to their childhood background (e.g., (1.28-1.25)/(1.28-1.00)*100=11%). The higher mortality among men with only one child is attributable in part to the higher likelihood of being sons of manual workers, which is associated with higher all-cause mortality.

Controlling for chronic conditions strongly affects the estimates among childless men, reducing them by approximately 20 per cent. The reduction reflects the fact that childless men are more likely to have a medical history of schizophrenia, intellectual disability or premature disability retirement than men with two children (Table 2). Controlling for chronic conditions has a barely noticeable effect on the estimates in the other parity groups.

Controls for living conditions in early adulthood has a large impact on the estimates in the case of men with no or only one child, in part because those in the former group are more likely to be never married and those in the latter group to be divorced than men with two children. The

reduction also relates to childless men with lower levels of education and income, and a higher likelihood of living in a poorly equipped dwelling. Controlling for living conditions in early adulthood renders the excess mortality among men with at least four children non-significant.

The excess mortality of men with no children and with only one child is reduced by approximately 50 per cent in the fully adjusted model for all men, after controlling for observed living conditions in childhood, chronic conditions, and living conditions in adulthood (Table 3, model 4 vs. model 1). Further controls for the timing of the first child reinforce the excess mortality among men with only one child (model 5). If men with only one child had had the child as early as those with two children had their first child, their mortality disadvantage would have been clearer, given the association between having a first child later in life and reduced male mortality.

Among the men, the findings from the fixed-effect models resemble those obtained from conventional Cox models (Figure 1): there is a significant increase in total mortality among those with no or only one child regardless of the unobserved characteristics shared by brothers, and observed chronic conditions and characteristics in adulthood.

All-cause mortality among women

With regard to women, hazards of death relative to those with two children is highest among the childless (HR = 1.75, 95% CI 1.59, 1.93) and the next highest among those with only one child (HR = 1.21, 95% CI 1.10, 1.33), independently of the year of birth. The mortality of women with three or at least four children is not significantly different from that of women with two children (Table 4).

Controlling for living conditions in childhood has barely noticeable effects on the estimates for women. Controls for chronic conditions, including intellectual disability, schizophrenia and disability retirement strongly affects the estimates among childless women, which decrease by approximately 35 per cent. The corresponding impact in the case of women with

one child is modest, but noticeable. Including further controls for living conditions in adulthood also affects the estimates: the excess mortality of women with one child reduces by approximately 17 per cent, and that of childless women by eight per cent. The stronger reduction in the former is attributable in part to divorce, which is more common among women with one child as opposed to two children. The reduction among childless women relates to never having married, which is associated with an elevated mortality risk. Childless women are not disadvantaged in terms of education or income, however.

The excess mortality of childless women is reduced by approximately 40 per cent in the fully adjusted model, and that of women with only one child by 30 per cent following controls for observed childhood characteristics, chronic conditions, and living conditions in early adulthood. Further controls for the timing of the first child have modest effects on the estimates. All-cause mortality among women with three or at least four children is not significantly different from that of women with two children in any of the models.

Among the women, the results from the fixed-effect Cox models are somewhat similar to those obtained from the conventional models (Figure 2). All-cause mortality is significantly higher among women with only one child than among those with two children, regardless of the unobserved characteristics shared by sisters and of all observed characteristics. However, the mortality of childless women differs from that of women with two children only at the 10-per-cent significance level.

Cardiovascular mortality among men

According to the conventional Cox model, childless men have the highest level of cardiovascular mortality, and men with at least four children and those with only one child the next highest, independently of the year of birth. Men with three children do not experience an elevated cardiovascular mortality relative to those with two children (Table 3, Model 1).

Controlling for childhood living conditions has relatively modest effects on the cardiovascular estimates, whereas controlling for chronic conditions has a large impact in the case of childless men, reducing them by approximately 20 per cent. The reduction relates mainly to a history of schizophrenia and disability retirement, which are more common among childless men, and strongly associated with cardiovascular mortality. Further controls for living conditions in early adulthood have large impacts on estimates among all parity groups with elevated mortality.

In the fully adjusted model for all men, controlling for observed living conditions in childhood, chronic conditions, and living conditions in early adulthood reduces the excess cardiovascular mortality among those with no or only one child by approximately 50 per cent, and among those with at least four children by 30 per cent. However, the reverse J-shaped relationship between the number of children and male mortality attributable to cardiovascular causes remains significant, independently of all the observed controls.

Other cause-specific mortality among men

Mortality attributable to violence and accidents is significantly higher among childless men than among those with two children, independent of the year of birth. In the case of alcohol-related causes, lung cancer and other cancers the risk is highest among childless men, followed by men with only one child. Controls for living conditions in childhood have a relatively modest effect on the cause-specific estimates. Controlling for chronic conditions has a relatively large impact on mortality estimates in the case of lung cancer, other cancers and violent and accidental causes among childless men, whereas no major changes are observable in the estimates of alcohol-related mortality. Further controls for living conditions in adulthood have a major impact on most of the cause-specific male estimates.

In the fully adjusted models, childless men still show higher mortality attributable to alcohol-related causes, accidents and violence, and lung cancer than men with two children. Men

with only one child have higher mortality attributable to alcohol-related causes and other cancers than those with two children, regardless of all the observed controls, including the timing of the first child. There is no difference in cause-specific mortality among men with three as opposed to two children in any of the models.

Cause-specific mortality among women

Mortality attributable to cardiovascular diseases, accidents and violence, breast cancer and other cancers is higher among childless women than among women with two children, regardless of the year of birth. Women with only one child have higher mortality attributable to cardiovascular disease and breast cancer than those with two children. Women with three children have higher mortality from breast cancer. Unexpectedly, women with at least four children face an increased risk of death from alcohol-related causes compared to those with two children.

Controls for living conditions in childhood have modest or no effects on the female estimates. On the other hand, including controls for chronic medical conditions has a relatively large impact on the estimated association between childlessness and mortality attributable to cardiovascular causes, accidents and violence, breast cancer and other cancers. Mortality attributable to accidental and violent causes is higher among childless women, partly because they are more likely to have a history of schizophrenia, which is strongly associated with the risk of dying from accidents and violence (HR = 3.74, 95% CI 2.15, 6.51). Controlled for chronic conditions renders the excess mortality observed in childless women non-significant.

Further controlling for living conditions in adulthood affects the cause-specific estimates. The excess mortality attributable to breast and other cancers among childless women is no longer significant, whereas the corresponding excess lung-cancer mortality becomes apparent. Further controlling for the timing of the first child renders the excess alcohol-related mortality

among women with at least four children non-significant (model 5). The excess cardiovascular mortality of women with no children or only one child nevertheless remains significant.

Discussion

Our study is among the few to examine cause-specific mortality by the individual number of children among men as well as women. Its novelty value is in controlling for a large set of living conditions in childhood with no recall bias, and for severe chronic conditions known to be associated with family formation and mortality. We demonstrate that, net of chronic conditions and observed living conditions in childhood and early adulthood, all-cause mortality is higher among men and women with no children or only one child than among those with two children. The overall mortality of women with at least four children is not different from that of women with two children in any of the models we estimated. Our findings among women thus yield no evidence of a trade-off between childbearing and female survival in the long term, as implied in the disposable soma theory (Kirkwood 1977; Kirkwood and Rose 1991). Furthermore, the results from the fixed-effect sibling models support the notion that all-cause mortality among parents of one child is higher than among their brothers and sisters with two children. This finding is similar to that reported earlier with regard to Swedish parents (Torssander 2013). The present study adds to the existing knowledge in showing that childless men face an increased risk of death compared to men with two children, independently of unobserved characteristics shared by brothers, and of observed medical and living conditions in adulthood.

Our results suggest a reversed J-shaped relationship between the number of children and male cardiovascular mortality, independently of all observed confounders. Cardiovascular mortality turns out to be the highest among men with no children, followed by men with at least four and men with only one child, and the lowest among men with two or three children. This finding is important given that cardiovascular causes are the leading causes of death among men.

On the other hand, there appears to be no significant excess cardiovascular mortality among women with four or more children. This finding among women is somewhat in line with the results of a Norwegian population study indicating no excess mortality among women with at least four children compared to those with two children. It has been suggested that family-friendly policies applied in Norway may buffer against stress related to childrearing (Grundy and Kravdal 2010). It is possible, however, that the higher cardiovascular mortality among Finnish men with at least four children relates to the stresses attendant on a large family size and an unhealthy lifestyle, against which the relatively generous North European family support gives no protection. This idea is reflected in a recent research report documenting that Swedish men with at least five biological children have higher cardiovascular mortality than those with two children, net of various socioeconomic confounders (Barclay and Kolk 2015). However, unlike our study, the Swedish study design does not allow controlling for the early timing of the first child and marital status, which could bias the relationship between high parity and male cardiovascular mortality.

Our results among men support the notion that the association between high numbers of offspring and cardiovascular disease is likely to relate to social and emotional factors and lifestyle rather than biological factors (Dekker and Schouten 1993). It is suggested in an earlier study that lifestyle factors associated with large families could lead to obesity and an increased risk of coronary heart disease (Lawlor et al. 2003). It is also documented that men with at least five children have less healthy body-mass indices than those with two children (Tamakoshi et al. 2011). It is therefore possible that health-damaging lifestyle patterns leading to obesity among men with large numbers of offspring offset the marginal health benefits of the social network their children comprise. This argument is in line with empirical evidence that does not clearly indicate an association between the frequency of contact with children and reduced mortality attributable to heart disease (Barefoot et al. 2005).

It is also suggested that child-rearing responsibilities provide an incentive to avoid health-damaging behaviour, and that the health effects of such behavioural patterns could become apparent later in life (Grundy and Kravdal 2008; Grundy and Kravdal 2010). Our results provide some support for this hypothesis. Alcohol-related mortality among childless men is approximately 70-per-cent higher, and almost 30 per cent higher among men with only one child, compared to men with two children, independently of all the observed controls. Conversely, there is no significant increase among women with no children or only one child. The analyses are conditional on having survived until the age of 50, and it is possible that childless women with the most severe alcohol dependence died at a younger age.

The findings among Finnish women and men thus differ, reflecting the results observed in a Norwegian study documenting higher alcohol-related mortality among those with no children or only one child regardless of gender (Grundy and Kravdal 2010). Unexpectedly, the alcohol-related mortality risk among women with at least four children is almost 80-per-cent higher than among those with two, regardless of the year of birth, chronic conditions, and observed living conditions in childhood and early adulthood. It is possible that high-parity Finnish women born in 1938-50 became worn out with the frequent pregnancies and constant childrearing, and developed harmful coping patterns. However, controlling for the early timing of the first child renders the association between high parity and alcohol-related female mortality non-significant.

Childless men and women have higher lung-cancer mortality than parents with two children, independently of all the observed controls. This is in line with the hypothesis of parental avoidance of health-damaging behaviour, and with a previous Norwegian study (Grundy and Kravdal 2010). Our study implies, however, that the excess mortality among men with only one child is apparent in midlife, but not in old age (Appendix 4).

In line with our expectations, controlling for a medical history of chronic conditions including schizophrenia, intellectual disability and disability retirement attenuates the total

mortality estimates among childless men and women. Despite its low prevalence in the general population, schizophrenia contributes strongly to explaining the association between childlessness and mortality attributable to various causes. This finding is in line with the results of a Swedish study indicating a higher rate of mortality attributable to both natural and unnatural causes among people with schizophrenia (Crump et al. 2013). The fact that its role is particularly important in our study in the analysis of female mortality attributable to accidental and violent causes makes a new contribution to research literature aimed at identifying health-selection effects. However and unexpectedly, a medical history of schizophrenia, intellectual disability or premature disability retirement does not help to explain the increased alcohol-related mortality of childless men. It is possible that our health measures do not capture the selective influences of alcohol dependence.

The present study is the first to illustrate the relatively modest effect of including controls for observed living conditions in childhood on the estimated association between the number of children and mortality, whereas controlling for living conditions in adulthood has a bigger impact, especially among men. We could not compare this result with any previous findings because no studies thus far examine this association among men after controlling for a large set of childhood living conditions. Henretta (2007) reports in a study on women from the United States that those with at least five children have higher mortality rates than those with two, and that controlled for the age, race, nativity and educational level of the father renders the excess mortality non-significant. Including the father's education and nativity (as indicators of childhood background) in the model does not distinguish their impact from that of the demographic characteristics, however.

Studying the relationship between the number of children and survival is not straightforward because both share common determinants. We were able to control for severe chronic conditions that may precede the decision to avoid childbearing, or involuntary childlessness. This sheds new light on research aimed at identifying the contribution of health-selection effects to

the association between the number of children and mortality. We used the principal diagnosis and three secondary diagnoses leading to hospital admission in order to capture the correct prevalence of intellectual disability and schizophrenia. We also measured the history of early disability retirement among the studied individuals. It is thus unlikely that we grossly overestimate the association between childlessness and all-cause mortality among men. The relationship is also clearly confirmed in fixed-effect sibling models allowing control for unobserved social and genetic characteristics common to brothers.

Unfortunately, our data do not include direct information on personality traits, health behaviour or attitudes towards family formation. However, according to a survey carried out at the beginning of the 1970s, the majority of 18–29-year-old Finnish men and women thought that two or three children was the best family size (Ritamies and Visuri 1975). It is thus possible that men who later lived up to this generally accepted family norm had personality traits that enabled them to achieve it. It is also possible that for men who were born in 1938-50 having two or three children was a marker of a normative family lifestyle that co-existed with other beneficial characteristics promoting cardiovascular health. The identification of causal links between the number of children and cause-specific mortality would nevertheless benefit from future studies on the mediating effects of health behaviour.

Although Nordic register-based data allow the linkage of fathers and children via personal identification codes, some children who were fathered before these codes were in use may not be included in our data. Children who were born in the 1950s and 1960s are linked to their father only if they lived with him in the late 1960s. However, this is unlikely to bias our results given that only five per cent of Finnish children were born out of wedlock in the 1950s and 1960s (Pitkänen and Jalovaara 2007). Moreover, some of these children may have lived with their father even if the parents were not legally married. Children born after the introduction of personal identification codes are systematically linked to both parents regardless of who they lived with.

More research is needed to examine the complex association between the number of children and cause-specific mortality among men in different social and cultural contexts in order to further elaborate on the underlying mechanisms. It would also be useful to find out whether the lowest cardiovascular mortality would be observed among men with two or three children in other social and cultural settings in which this set-up is not a family norm. Furthermore, assessing the contribution of a spouse's characteristics in modifying the association between fertility and mortality offers promise as a direction for future study.

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Table 1. The distribution (%) of childhood characteristics by the number of children, Finnish men and women born in 1938-50 and alive at the age of 50, *N* = 87,573

	Men (N=44,800) Number of children						**Women** (N=42,773) Number of children					
	0	1	2	3	4+	All	0	1	2	3	4+	All
Family type												
Both parents	88	89	89	89	89	89	89	89	90	89	86	89
Only mother	8	7	7	6	6	7	7	7	7	7	8	7
Only father	1	1	1	1	1	1	1	1	1	1	1	1
No parents	4	3	3	3	4	3	3	3	3	3	5	3
Number of siblings												
None	14	16	15	12	12	14	15	15	14	12	11	14
One	23	26	26	24	21	25	26	27	25	23	18	25
Two to three	34	34	36	36	34	35	34	34	36	36	34	35
Four or more	25	21	21	24	28	23	22	21	22	25	32	23
Unknown	4	3	3	3	4	3	3	3	3	3	5	3
Parental education												
Less than primary school	21	17	16	17	20	18	17	18	18	19	25	19
Primary school	71	73	73	71	70	72	70	73	73	71	68	72
More than primary school	8	9	11	12	10	10	13	9	9	10	7	10
Occupation of family head												
Independent farmer	31	25	28	30	33	29	29	27	29	31	34	30
Other self-employed/employer	8	8	9	10	9	9	8	9	8	8	9	8
Professional/manager	13	15	17	16	14	15	19	15	15	14	11	15
Manual worker	46	50	45	43	43	46	42	48	46	45	44	45
Unknown	2	1	1	1	1	1	2	2	1	1	2	1
Home ownership												
Owner	61	56	59	61	63	59	58	58	59	62	67	60
Renter	22	26	24	22	21	24	24	25	24	22	19	23
Other	8	9	9	9	8	9	10	9	9	8	7	9
Unknown	9	9	8	8	9	8	8	8	8	8	7	8
Central heating												
Yes	9	10	11	10	9	10	12	9	10	9	7	10
No	90	89	88	89	90	89	87	90	89	90	92	89
Unknown	1	1	1	1	1	1	1	1	1	1	1	1
Piped water and sewer												
Yes	16	19	21	20	17	19	23	18	19	18	14	19
No	82	80	78	79	82	80	76	81	79	81	86	80
Unknown	1	1	1	1	1	1	1	1	1	1	1	1
Persons in household per room												
less than 2.00	32	33	38	37	32	35	39	34	35	34	29	35
2.00-3.00	42	44	42	40	43	42	41	43	42	42	41	42
3.00 and over	25	22	20	21	24	22	20	21	21	23	29	22
Unknown	2	1	1	1	1	1	1	1	1	1	1	1
All	100	100	100	100	100	100	100	100	100	100	100	100
N	8600	8128	16636	7865	3571	44800	6120	8541	16710	7777	3625	42773

Source: Census 1950 longitudinal data file from Statistics Finland and the National Institute for Health and Welfare

Table 2. The distribution (%) of chronic conditions and characteristics in early adulthood by the number of children, Finnish men and women born in 1938-50 and alive at the age of 50, *N* = 87,573

	Men (N=44,800) Number of children						**Women** (N=42,773) Number of children					
	0	1	2	3	4+	All	0	1	2	3	4+	All
Schizophrenia												
Yes	6	1	1	-	-	2	5	2	1	1	1	2
No	94	99	99	-	-	98	95	98	99	99	99	98
Intellectual disabilities												
Yes	2	-	-	-	-	1	2	-	-	-	-	1
No	98	-	-	-	-	100	98	-	-	-	-	100
Premature disability pension												
Yes	8	1	1	1	1	2	7	1	1	1	1	2
No	92	99	99	99	99	98	93	99	99	99	99	98
Education												
basic or less	59	49	43	45	51	48	42	50	48	52	63	50
secondary	27	31	30	28	28	29	30	30	30	28	23	29
tertiary	14	20	27	27	22	23	27	20	22	20	14	22
Marital status												
Married	18	73	87	88	87	72	26	71	88	89	89	76
Never married	76	16	7	5	4	21	68	15	4	2	1	15
Divorced/separated	3	9	4	5	7	5	4	12	6	6	7	7
Widowed/unknown	3	2	2	2	2	2	2	2	2	3	3	2
Annual income (1000s in euros of 2012)												
0-9	33	12	9	12	16	15	26	27	41	52	68	40
10-19	28	23	20	21	25	23	42	49	40	34	25	40
20-29	28	42	42	38	35	38	25	20	15	11	6	16
30+	11	22	29	29	25	24	7	4	3	3	2	3
Home ownership												
Owner	58	53	58	56	54	56	50	54	60	59	59	57
Renter	21	27	23	24	26	24	28	27	22	23	24	24
Other	8	13	14	14	13	12	12	13	13	13	12	13
Unknown	13	8	5	7	7	8	10	5	4	5	6	6
Level of equipment in a dwelling												
Well equipped	44	67	74	72	65	66	67	75	77	72	62	73
Poorly equipped	44	26	21	22	29	27	24	21	19	23	33	22
Unknown	11	7	5	6	7	7	9	5	4	5	5	5
Timing of the first child												
< 20 years		2	3	5	7	4		8	13	21	32	15
20-24 years		29	39	48	55	40		39	50	53	53	48
25-29 years		34	40	35	30	36		28	29	22	13	26
30-34 years		19	13	9	6	13		14	7	4	2	8
at least 35 years		16	5	3	2	7		10	2	1	0	3
All	100	100	100	100	100	100	100	100	100	100	100	100
N	8600	8128	16636	7865	3571	44800	6120	8541	16710	7777	3625	42773

(-) Prevalence not presented on account of the small numbers and the privacy policy of Statistics Finland and the National Institute for Health and Welfare.
Source: As for Table 1.

Table 3. The association between the number of children and all-cause and cause-specific mortality at ages 50-72 (hazard ratios and 95% confidence intervals), Finnish men born in 1938-50, *N* = 44,800

Cause of death	Men (N=44,800) Number of children									
	0		1		2 (ref.)	3		4+		
All causes	Model									
(deaths 7094)	1	**2.00**	(1.88-2.13)	**1.28**	(1.19-1.37)	1.00	1.00	(0.93-1.07)	**1.17**	(1.07-1.29)
	2	**1.96**	(1.85-2.09)	**1.25**	(1.17-1.34)	1.00	1.00	(0.93-1.08)	**1.18**	(1.07-1.29)
	3	**1.77**	(1.66-1.89)	**1.24**	(1.16-1.33)	1.00	1.00	(0.93-1.08)	**1.18**	(1.07-1.29)
	4	**1.48**	(1.37-1.61)	**1.13**	(1.06-1.21)	1.00	0.98	(0.91-1.05)	1.09	(0.99-1.20)
	5			**1.20**	(1.12-1.29)	1.00	0.95	(0.88-1.03)	1.05	(0.95-1.15)
Cardiovascular diseases										
(2583 deaths)	1	**2.19**	(1.98-2.42)	**1.28**	(1.14-1.44)	1.00	1.02	(0.90-1.15)	**1.41**	(1.22-1.63)
	2	**2.13**	(1.93-2.36)	**1.25**	(1.12-1.41)	1.00	1.02	(0.90-1.15)	**1.40**	(1.21-1.62)
	3	**1.91**	(1.72-2.12)	**1.25**	(1.11-1.40)	1.00	1.02	(0.90-1.15)	**1.40**	(1.21-1.62)
	4	**1.53**	(1.35-1.75)	**1.13**	(1.01-1.28)	1.00	1.00	(0.88-1.13)	**1.28**	(1.10-1.49)
	5			**1.20**	(1.06-1.35)	1.00	0.97	(0.86-1.10)	**1.24**	(1.07-1.44)
Alcohol-related causes										
(822 deaths)	1	**1.91**	(1.60-2.28)	**1.39**	(1.14-1.69)	1.00	0.90	(0.72-1.13)	1.05	(0.79-1.40)
	2	**1.89**	(1.58-2.26)	**1.34**	(1.10-1.63)	1.00	0.92	(0.73-1.15)	1.06	(0.80-1.42)
	3	**1.88**	(1.56-2.25)	**1.34**	(1.10-1.63)	1.00	0.92	(0.73-1.15)	1.06	(0.80-1.42)
	4	**1.73**	(1.38-2.17)	1.18	(0.97-1.44)	1.00	0.89	(0.71-1.11)	0.95	(0.71-1.27)
	5			**1.27**	(1.03-1.56)	1.00	0.85	(0.68-1.06)	0.88	(0.66-1.19)
Accidents & violence										
(922 deaths)	1	**1.96**	(1.66-2.31)	**1.21**	(1.00-1.46)	1.00	0.88	(0.71-1.09)	0.95	(0.72-1.26)
	2	**1.89**	(1.60-2.23)	1.18	(0.98-1.43)	1.00	0.89	(0.72-1.10)	0.95	(0.72-1.25)
	3	**1.75**	(1.47-2.09)	1.18	(0.97-1.42)	1.00	0.89	(0.72-1.10)	0.95	(0.72-1.25)
	4	**1.43**	(1.16-1.76)	1.04	(0.86-1.26)	1.00	0.87	(0.70-1.07)	0.85	(0.64-1.13)
	5			1.12	(0.92-1.36)	1.00	0.85	(0.69-1.05)	0.82	(0.62-1.09)
Lung cancer										
(543 deaths)	1	**2.04**	(1.63-2.55)	**1.33**	(1.03-1.70)	1.00	1.16	(0.89-1.50)	1.26	(0.91-1.77)
	2	**1.97**	(1.57-2.46)	1.28	(1.00-1.64)	1.00	1.15	(0.89-1.49)	1.24	(0.89-1.73)
	3	**1.72**	(1.36-2.18)	1.27	(0.99-1.63)	1.00	1.15	(0.89-1.49)	1.24	(0.89-1.73)
	4	**1.57**	(1.16-2.14)	1.17	(0.90-1.50)	1.00	1.12	(0.86-1.45)	1.14	(0.82-1.59)
	5			1.27	(0.97-1.65)	1.00	1.07	(0.83-1.38)	1.05	(0.76-1.46)
Other cancers										
(1326 deaths)	1	**1.27**	(1.09-1.48)	**1.22**	(1.05-1.42)	1.00	0.98	(0.83-1.07)	1.03	(0.83-1.28)
	2	**1.29**	(1.11-1.49)	**1.22**	(1.05-1.42)	1.00	0.99	(0.84-1.08)	1.05	(0.85-1.31)
	3	**1.23**	(1.06-1.43)	**1.22**	(1.05-1.41)	1.00	0.98	(0.84-1.08)	1.05	(0.85-1.31)
	4	1.09	(0.91-1.31)	**1.17**	(1.00-1.36)	1.00	0.98	(0.83-1.05)	1.03	(0.83-1.29)
	5			**1.22**	(1.04-1.42)	1.00	0.96	(0.82-1.03)	1.02	(0.82-1.28)

Bold: $p < 0.05$
Model 1: controlling for the year of birth
Model 2: Model 1 + living conditions in childhood
Model 3: Model 2 + chronic conditions, including intellectual disability, schizophrenia, and early disability retirement
Model 4: Model 3 + living conditions in early adulthood
Model 5 (for parents): Model 4 + timing of the first child
Source: As for Table 1.

Table 4. The association between the number of children and all-cause and cause-specific mortality at ages 50-72 (hazard ratios and 95% confidence intervals), Finnish women born in 1938-50, *N* = 42,773

Cause of death		Women (N=42,773) Number of children								
		0		1		2 (ref.)	3		4+	
All causes	Model									
(deaths 3170)	1	**1.75**	(1.59-1.93)	**1.21**	(1.10-1.33)	1.00	0.95	(0.85-1.05)	1.13	(0.99-1.29)
	2	**1.75**	(1.59-1.93)	**1.20**	(1.09-1.33)	1.00	0.95	(0.85-1.05)	1.12	(0.98-1.27)
	3	**1.49**	(1.34-1.65)	**1.18**	(1.07-1.30)	1.00	0.95	(0.85-1.05)	1.12	(0.98-1.27)
	4	**1.44**	(1.27-1.64)	**1.15**	(1.04-1.27)	1.00	0.92	(0.83-1.03)	1.04	(0.91-1.19)
	5			**1.16**	(1.04-1.28)	1.00	0.91	(0.81-1.01)	1.00	(0.87-1.14)
Cardiovascular diseases										
(703 deaths)	1	**2.09**	(1.71-2.56)	**1.37**	(1.11-1.69)	1.00	0.99	(0.78-1.25)	1.28	(0.97-1.68)
	2	**2.10**	(1.71-2.58)	**1.36**	(1.10-1.68)	1.00	0.97	(0.77-1.23)	1.21	(0.92-1.59)
	3	**1.71**	(1.38-2.14)	**1.33**	(1.08-1.64)	1.00	0.97	(0.77-1.23)	1.21	(0.92-1.59)
	4	**1.53**	(1.16-2.02)	**1.28**	(1.03-1.59)	1.00	0.92	(0.73-1.17)	1.05	(0.80-1.39)
	5			**1.33**	(1.06-1.66)	1.00	0.87	(0.69-1.11)	0.96	(0.72-1.27)
Alcohol-related causes										
(215 deaths)	1	1.20	(0.80-1.81)	1.36	(0.94-1.95)	1.00	1.03	(0.69-1.55)	**1.75**	(1.13-2.70)
	2	1.17	(0.77-1.77)	1.34	(0.93-1.93)	1.00	1.04	(0.69-1.56)	**1.72**	(1.11-2.67)
	3	1.20	(0.79-1.84)	1.34	(0.93-1.94)	1.00	1.04	(0.69-1.56)	**1.72**	(1.11-2.67)
	4	1.18	(0.71-1.96)	1.18	(0.82-1.71)	1.00	1.07	(0.71-1.62)	**1.80**	(1.13-2.86)
	5			1.34	(0.92-1.95)	1.00	0.97	(0.64-1.47)	1.50	(0.93-2.43)
Accidents & violence										
(263 deaths)	1	**1.49**	(1.06-2.08)	1.12	(0.81-1.56)	1.00	0.83	(0.57-1.20)	0.94	(0.58-1.50)
	2	**1.47**	(1.05-2.06)	1.11	(0.80-1.55)	1.00	0.83	(0.57-1.21)	0.94	(0.58-1.52)
	3	1.18	(0.82-1.70)	1.07	(0.77-1.49)	1.00	0.83	(0.57-1.21)	0.94	(0.58-1.52)
	4	0.97	(0.63-1.49)	0.96	(0.69-1.35)	1.00	0.85	(0.58-1.24)	0.97	(0.60-1.56)
	5			1.03	(0.73-1.44)	1.00	0.80	(0.55-1.17)	0.87	(0.54-1.42)
Lung cancer										
(208 deaths)	1	1.41	(0.95-2.11)	1.12	(0.77-1.65)	1.00	1.03	(0.69-1.54)	1.46	(0.92-2.30)
	2	1.41	(0.94-2.11)	1.13	(0.77-1.66)	1.00	1.02	(0.68-1.53)	1.41	(0.89-2.23)
	3	1.37	(0.91-2.07)	1.12	(0.76-1.65)	1.00	1.02	(0.68-1.53)	1.41	(0.89-2.23)
	4	**1.84**	(1.10-3.09)	1.05	(0.71-1.56)	1.00	1.01	(0.67-1.50)	1.35	(0.84-2.15)
	5			1.05	(0.70-1.60)	1.00	0.95	(0.63-1.43)	1.17	(0.73-1.89)
Breast cancer										
(355 deaths)	1	**1.73**	(1.28-2.33)	**1.48**	(1.11-1.97)	1.00	**1.39**	(1.03-1.86)	0.85	(0.53-1.34)
	2	**1.70**	(1.26-2.29)	**1.49**	(1.12-1.98)	1.00	**1.40**	(1.04-1.88)	0.91	(0.58-1.45)
	3	**1.58**	(1.17-2.15)	**1.48**	(1.11-1.97)	1.00	**1.40**	(1.04-1.88)	0.91	(0.57-1.45)
	4	1.27	(0.85-1.88)	**1.45**	(1.08-1.95)	1.00	**1.41**	(1.05-1.91)	0.93	(0.58-1.49)
	5			1.33	(0.98-1.81)	1.00	**1.48**	(1.10-2.01)	1.03	(0.64-1.67)
Other cancers										
(907 deaths)	1	**1.30**	(1.08-1.57)	1.02	(0.85-1.22)	1.00	0.85	(0.69-1.05)	1.03	(0.81-1.30)
	2	**1.30**	(1.08-1.57)	1.02	(0.85-1.22)	1.00	0.86	(0.70-1.05)	1.04	(0.81-1.32)
	3	**1.24**	(1.03-1.51)	1.01	(0.84-1.21)	1.00	0.86	(0.70-1.05)	1.04	(0.81-1.32)
	4	1.23	(0.96-1.58)	1.02	(0.85-1.23)	1.00	0.84	(0.69-1.03)	0.99	(0.77-1.26)
	5			1.02	(0.84-1.24)	1.00	0.84	(0.69-1.01)	0.98	(0.76-1.27)

Bold: $p < 0.05$

Model 1: controlling for the year of birth

Model 2: Model 1 + living conditions in childhood

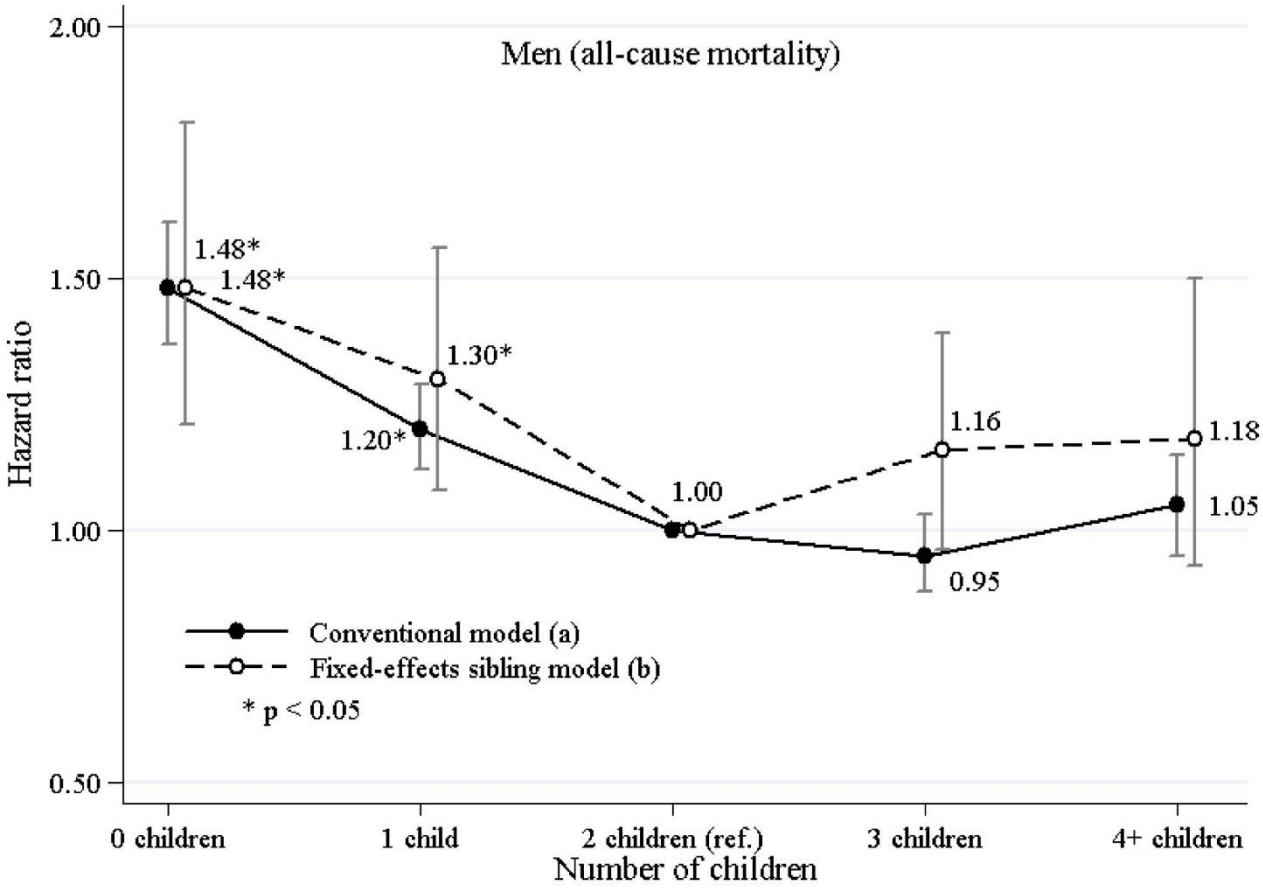
Model 3: Model 2 + chronic conditions, including intellectual disability, schizophrenia, and early disability retirement

Model 4: Model 3 + living conditions in early adulthood

Model 5 (for parents): Model 4 + timing of the first child

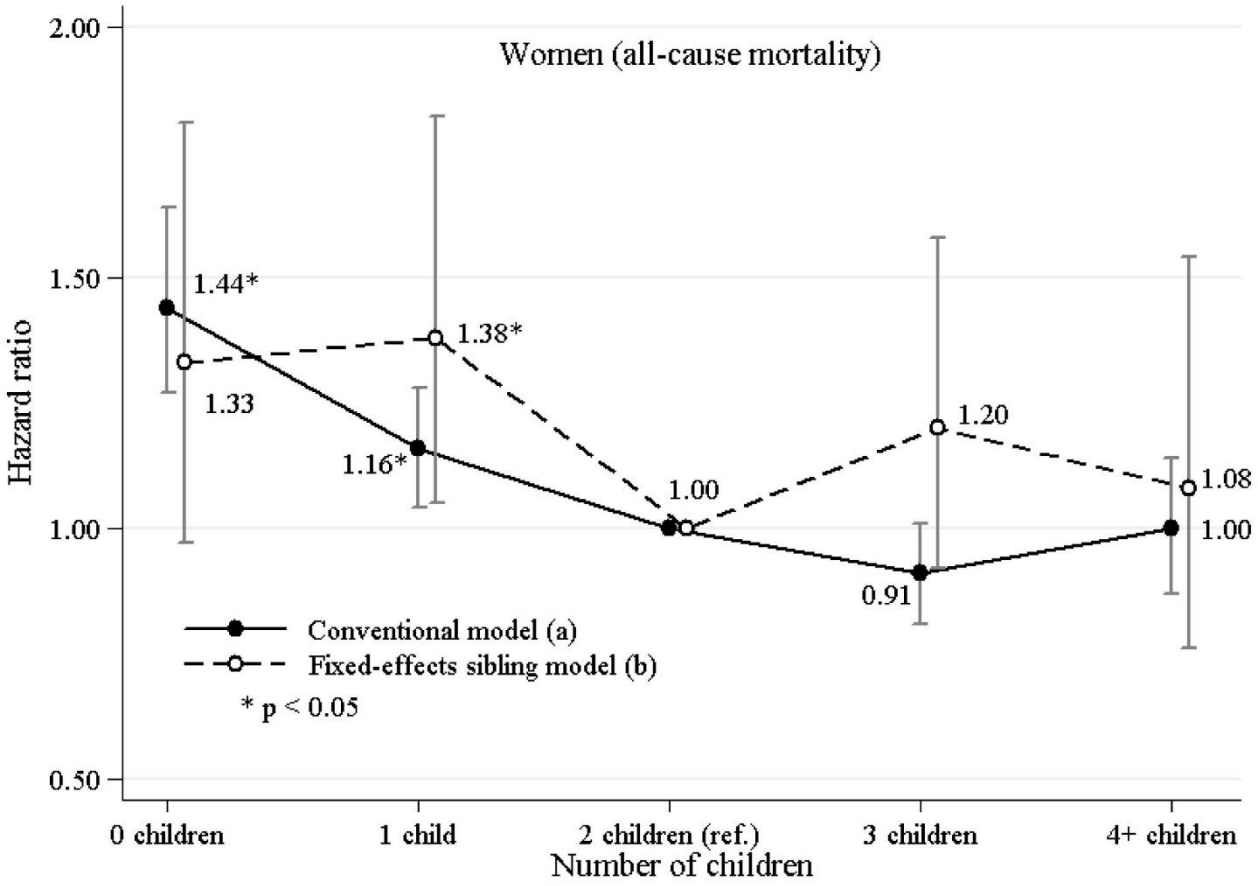
Source: As for Table 1.

Figure 1. HR and 95 per cent CI of all-cause mortality at ages 50-72 in relation to the number of children (two children: HR = 1), Finnish men born in 1938-1950, conventional and fixed-effects Cox models, *N* = 44,800 and *N* = 7,032



(a) Conventional model controls for observed characteristics, including the year of birth, chronic conditions, characteristics in childhood and early adulthood, and for the parents also the timing of the first child.
(b) Fixed-effects sibling model controls for observed characteristics and unobserved characteristics shared by brothers.
Source: As for Table 1.

Figure 2. HR and 95 per cent CI of all-cause mortality at ages 50-72 in relation to the number of children (two children: HR = 1), Finnish women born in 1938-1950, conventional and fixed-effects Cox models, *N* = 42,773 and *N* = 3,283



(a) Conventional model controls for observed characteristics, including the year of birth, chronic conditions, characteristics in childhood and early adulthood, and for the parents also the timing of the first child.
(b) Fixed-effects sibling model controls for observed characteristics and unobserved characteristics shared by sisters.
Source: As for Table 1.